

Changes in Intraocular Pressure due to Surgical Positioning

Studying Potential Risk for Postoperative Vision Loss

Kristina S. Walick, MD,* John E. Kragh, Jr, MD,† John A. Ward, PhD,‡
and John J. Crawford, MD*

Study Design. Parallel group design.

Objective. Compare the intraocular pressure responses in the prone flat *versus* prone Trendelenburg's position.

Summary of Background Data. Postoperative vision loss (PVL) complicates approximately 0.05% of spine surgeries. Prone positioning is considered a risk factor because it increases intraocular pressure, which may decrease perfusion pressure to the optic nerve (perfusion pressure = mean arterial pressure – intraocular pressure [IOP]). The prone Trendelenburg's position is often used during spine surgery; however, its effect on optic nerve perfusion is unknown. The purpose of this study is to compare the IOP responses in the prone flat *versus* prone Trendelenburg's positions to determine if prone Trendelenburg's position also risks PVL.

Methods. Twenty subjects randomized into 2 groups. Group 1 lay in the prone flat position (0°). Group 2 lay in the prone Trendelenburg's position (-7°). IOPs were measured with a hand-held applanation tonometer while seated, 1 minute after assuming the group's position (Time 0), and at 10-minute intervals for 60 minutes.

Results. The differences in mean IOPs with respect to positions and time were significant ($P = 0.0001$, $P = 0.000$). There was a significant difference between sitting and all other times for both groups. In Group 1, there was a significant difference in IOP between Time 0 and all other times prone flat ($P < 0.05$). In Group 2, there was a significant difference in IOP between Time 0 all other times prone Trendelenburg ($P < 0.05$).

Conclusion. IOP increases in the prone Trendelenburg's position, and when combined with other factors, may be a risk factor for PVL. The pathophysiology is discussed and suggestions for clinicians are made.

Key words: intraocular pressure, prone Trendelenburg, postoperative vision loss. *Spine* 2007;32:2591–2595

Vision loss after spine surgery occurs in approximately 1 in 500 to 1 in 1000 procedures, or 1 case per 100 spine surgeons per year.^{1–5} Until recently, authors attributed vision loss to increased intraocular pressure (IOP) secondary to inadvertent orbital compression or an ill fitting headrest during surgery.^{6–12} The contribution of poor head positioning was challenged by evidence from cases where external compression of the orbits was clearly not a factor.^{13–15} Recent studies now state that risk factors may be hypotension, anemia, obesity, peripheral vascular disease, and middle-aged men.^{1,3,4,12,16–25} Yet there are cases where none of these risk factors was present.^{12,14} The one risk factor present in every case is prone position. Prone positioning is considered a risk factor because it increases IOP, which decreases perfusion pressure to the optic nerve. This occurs because perfusion pressure equals mean arterial pressure minus IOP.²⁶ Prone Trendelenburg's position (or head-down tilt) is occasionally used during spine surgery while repairing a dural tear or to enhance exposure. However, the effect of this surgical position on IOP and perfusion to the optic nerve during spine surgery is not known. Moreover, most surgeons and anesthesiologists do not record the degree of table tilt used during surgery nor do they monitor the duration in position. We chose to study the timing and magnitude of IOP in the prone Trendelenburg's position to determine if this position contributes to decreased perfusion pressure and possibly risks postoperative vision loss. The purpose of our study is to determine if normal volunteers have increased IOP in the prone Trendelenburg's position compared with the prone flat position over a 1-hour study time.

■ Materials and Methods

Our target population included healthy men and women volunteers older than age 18 years. Volunteers were included if they had no history of glaucoma, eye trauma, injury or surgery to either eye, including Laser-Assisted In Situ Keratomileusis and photorefractive keratectomy. Patients with one of the above conditions or allergy to topical anesthetics were excluded.

After approval by our Institutional Review Board, informed consent was obtained from 20 healthy volunteers 25 to 40 years of age. Subjects were randomly divided into 2 groups designated to lay either prone flat or prone Trendelenburg. The eyes were anesthetized with 0.05% tetracaine topical anesthetic while subjects were seated and reanesthetized intermittently as needed for comfort. Both groups were seated for an initial baseline measurement of IOP recording in order to determine the similarity of IOP in the 2 groups before position

From the *Department of Orthopaedics and Rehabilitation, Brooke Army Medical Center, †United States Army Institute of Surgical Research, and ‡Department of Clinical Investigation, Fort Sam Houston, TX.

Acknowledgment date: August 25, 2006. First revision date: March 24, 2007. Acceptance date: April 30, 2007.

The manuscript submitted does not contain information about medical device(s)/drug(s).

No funds were received in support of this work. No benefits in any form have been or will be received from a commercial party related directly or indirectly to the subject of this manuscript.

The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or reflecting the views of the Department of Defense or US Government. The authors are employees of the US government. This work was prepared as part of their official duties and, as such there is no copyright to be transferred.

Address correspondence and reprint requests to Kristina S. Walick, MD, 134 Lamont Ave, San Antonio, TX 78209; E-mail: Kristina.walick@amedd.army.mil

Report Documentation Page			Form Approved OMB No. 0704-0188		
Public reporting burden for the collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to Washington Headquarters Services, Directorate for Information Operations and Reports, 1215 Jefferson Davis Highway, Suite 1204, Arlington VA 22202-4302. Respondents should be aware that notwithstanding any other provision of law, no person shall be subject to a penalty for failing to comply with a collection of information if it does not display a currently valid OMB control number.					
1. REPORT DATE 01 NOV 2007	2. REPORT TYPE N/A	3. DATES COVERED -			
4. TITLE AND SUBTITLE Changes in intraocular pressure due to surgical positioning: studying potential risk for postoperative vision loss			5a. CONTRACT NUMBER		
			5b. GRANT NUMBER		
			5c. PROGRAM ELEMENT NUMBER		
6. AUTHOR(S) Walick K. S., Kragh Jr. J. E., Ward J. A., Crawford J. J.,			5d. PROJECT NUMBER		
			5e. TASK NUMBER		
			5f. WORK UNIT NUMBER		
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) United States Army Institute of Surgical Research, JBSA Fort Sam Houston, TX 78234			8. PERFORMING ORGANIZATION REPORT NUMBER		
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)			10. SPONSOR/MONITOR'S ACRONYM(S)		
			11. SPONSOR/MONITOR'S REPORT NUMBER(S)		
12. DISTRIBUTION/AVAILABILITY STATEMENT Approved for public release, distribution unlimited					
13. SUPPLEMENTARY NOTES					
14. ABSTRACT					
15. SUBJECT TERMS					
16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT UU	18. NUMBER OF PAGES 5	19a. NAME OF RESPONSIBLE PERSON
a. REPORT unclassified	b. ABSTRACT unclassified	c. THIS PAGE unclassified			

Table 1. Descriptive Statistics for Intraocular Pressure Measurements

Group	Position	Time (min)	Mean IOP (mm Hg)	95% CI
Prone flat (N = 10)	Seated Prone flat (0°)	0	19.3 ± 2.9	13.6–25.0
		0	25.8 ± 4.4	17.2–34.4
		10	29.7 ± 4.1	21.6–37.8
		20	29.6 ± 6.3	17.2–42.0
		30	32.0 ± 5.0	22.2–41.8
		40	30.0 ± 4.8	20.6–39.4
		50	30.8 ± 4.1	22.8–38.7
		60	31.1 ± 3.4	24.4–37.7
Prone Trendelenburg (N = 10)	Seated Prone Trendelenburg (-7°)	0	21.4 ± 4.5	12.5–30.2
		0	31.1 ± 2.8	25.6–36.6
		10	36.2 ± 4.0	28.3–44.1
		20	36.9 ± 4.9	27.2–46.6
		30	36.8 ± 6.2	24.7–48.9
		40	36.4 ± 2.6	31.4–41.4
		50	35.6 ± 5.3	25.1–46.0
		60	37.5 ± 4.6	28.4–46.5

There was a significant difference between mean IOP in the sitting position and all other mean IOPs from 0 to 60 min ($P < 0.001$, S-N-K test). There was a significant difference between mean IOP at 0 min in the prone or prone Trendelenburg's position and all other mean IOPs from 10 to 60 min ($P < 0.001$, S-N-K test). There was no significant difference between IOPs from 10 through 60 min ($P = 1.000$, S-N-K test).

IOP indicates intraocular pressure; CI, confidence interval.

change. All measurements were made with a hand-held applanation tonometer by an ophthalmologist (Tonopen, Medtronic Solan, Jacksonville, FL) in the left eye.²⁷ Next, the subjects lay prone in their designated test positions on a well-padded operating room bed without bolsters. All faces were carefully placed in a horseshoe headrest (Quantum 3080 SP, Steris Corp., Montgomery, LA) to prevent extraocular pressure. This setup was chosen to facilitate exposure to the eyes so that IOP measurements could be made without moving the head. Neck flexion and extension were minimized such that the head was in line with the thorax.

The prone flat group laid prone on a table with 0° incline so that the eyes were at the level of the heart. The prone Trendelenburg group laid in the prone Trendelenburg's position with the head of the table inclined to -7° so that the eyes were below the level of the heart. The incline angle was measured with a goniometer. IOP was measured within 1 minute of assuming the test positions in both groups (designated as Time 0 in position). Measurements were then made at 10 minute intervals, for 60 minutes.

The hand-held applanation tonometer has been shown to be a reproducible and reliable method to measure IOP.^{27,28} After it contacts the cornea one time, the unit displays the average of 4 independent readings along with the standard deviation and confidence interval. A minimum of 2 contacts with measure-

ments within the 5% confidence interval were obtained and then averaged for each individual.

Our statistical analysis was done with Sigma Stat version 3.11 (Systat Software, Inc., Point Richmond, CA) using a 2-factor analysis of variance (position, time) with repeated measures on one factor (time) to determine if there was a difference in mean IOP with respect to position and time between groups. Next, we used a Student-Newman-Keuls (S-N-K) *post hoc* test to determine the difference in mean IOP with respect to time within each group. P levels less than 0.05 were considered statistically significant.

■ Results

There was no significant difference between group mean IOPs while seated ($P > 0.312$, S-N-K test, Table 1). The differences in mean IOPs with respect to both position and time were significant ($P = 0.001$ and $P < 0.001$, respectively, Figure 1). There was a statistically significant difference in mean IOP in the sitting position between all other mean IOPs from 0 to 60 minutes ($P < 0.001$, S-N-K test). There was a statistically significant difference in mean IOP between Time 0 in the prone flat

Mean and Standard Deviation of Intraocular Pressure

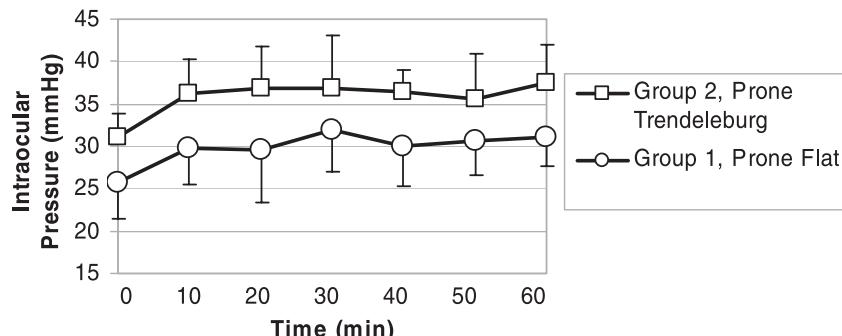


Figure 1. The difference between group means is statistically significant at every time.

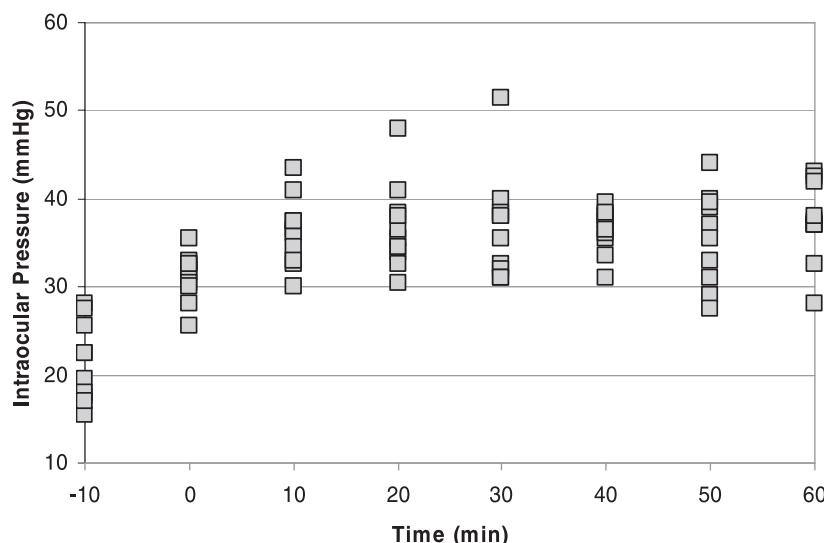
IOP Prone Flat Group and Prone Trendelenburg Groups**Intraocular Pressure Prone Trendelenburg**

Figure 2. Intraocular pressures are greater than 40 mm Hg at every time measured after 10 minutes in position and measurements are as high as 51 mm Hg. Pressure measured while sitting is designated as time -10 .

position and all other times prone flat ($P < 0.05$). In the prone Trendelenburg group, there was a statistically significant difference in mean IOP between Time 0 in prone Trendelenburg's position and all other times prone Trendelenburg ($P < 0.05$). There was no significant interaction between position and time ($P = 0.351$), meaning that the pressure trends maintain the same difference throughout the study time period.

The 5.4 mm Hg difference in mean IOP between prone Trendelenburg and prone flat positions was statistically significant ($P = 0.001$). The mean IOP difference between groups at Time 0 (5.3 mm Hg, $P = 0.005$) was statistically significant and remained statistically significant at Time 60 minutes (6.4 mm Hg, $P = 0.002$).

■ Discussion

The idea of IOP being associated with increases in the prone position is not new, but the current study is original in that it is the first to compare IOPs of subjects in the prone flat *versus* Trendelenburg position for the extended duration of 1 hour. The main finding of this study is that surgical positions and time in surgical positions elevate IOP to levels that have been thought to risk ischemic optic neuropathy^{26,29} (Figure 2).

The mean difference IOP between both groups may be clinically relevant (Figure 1). The blood flow in the optic nerve head remains relatively constant despite changes in IOP due to an increase in mean arterial pressure as a result of autoregulation in the vascular system.^{29,30} However, beyond a certain point, autoregulation has been shown to fail. Recent data in human volunteers indicate that blood flow in the optic nerve head is constant until ocular pressures reach 40 mm Hg.³⁰ In monkeys, sustained IOPs >50 mm Hg for 180 minutes have induced optic neuropathy.³¹⁻³³ Additionally, vascular endothelial dysfunction interferes with autoregulation

and is known to occur in arterial hypertension, diabetes, atherosclerosis, hypercholesterolemia, aging, and other unknown causes.²⁶ Therefore, in select patients, a small elevation of IOP above a critical level may tip the scale. Our 1-hour study results suggest in these patients longer time in prone Trendelenburg may be harmful.

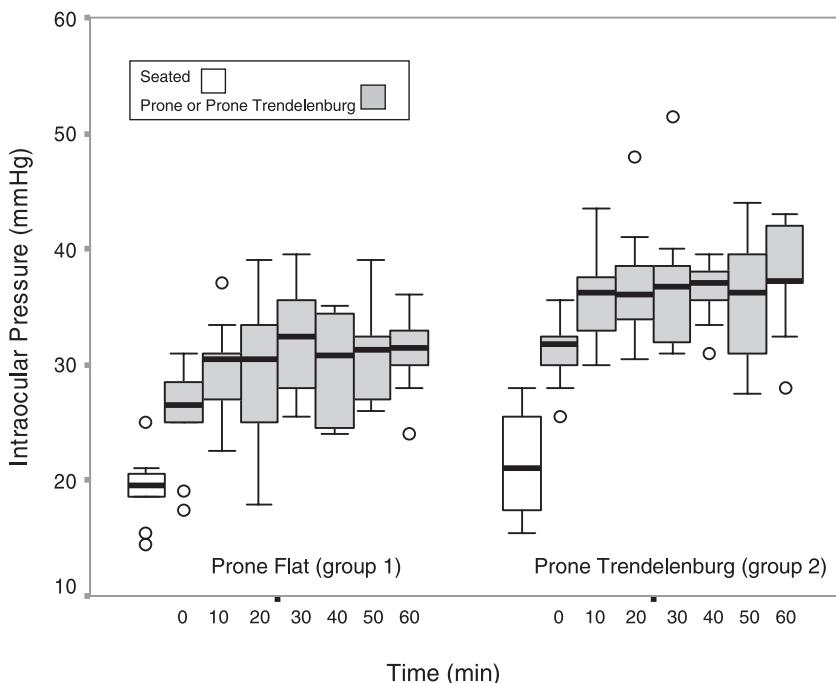
The increase of IOP with time in position advances and confirms prior similar findings (Figure 3). Similar sustained increases in IOP over time have been reported in animal studies as well as humans and have been shown to have negative effects.^{27,29,32-35} Draeger and Hanke found that IOP in supine Trendelenburg subjects reached a maximum at 15 minutes and then reached a plateau after 45 minutes.³⁶ Cheng *et al* found increased time in the prone position in anesthetized patients correlated with the increased IOP measured at the end of the case.^{37,38}

The effects of prone positioning and Trendelenburg positioning are additive.³⁹ Turning a patient from supine to prone, abdominal compression, and tilting the head below the heart each increases IOP independently.^{4,36,39,40-43} Prone Trendelenburg combines these, giving the higher IOP measurements recorded in our study.

Subjects in prone Trendelenburg had more facial discomfort over the duration of the experiment than those prone flat. These findings support prior clinical findings.^{29,34,35,42} The subjects in prone Trendelenburg complained more of headache, nasal congestion, and a sensation of pressure behind the eyes, which steadily worsened through the testing.

There are several weaknesses of our study. We tested normal volunteers, not the patients who undergo spine surgery. Patients are more likely to have one or more identifiable risk factors of ischemic optic neuropathy already. We did not conduct the study during surgery, so patients were not anesthetized. General anesthesia has been shown to decrease IOP.⁴⁴ While testing subjects we

Figure 3. The data are represented as median (dark line) within 25th and 75th percentiles (boxes). The flags represent the largest and smallest observed values that are not outliers. Outliers are values more than 1.5 box lengths from the quartile and are represented as circles. Mean IOP differences between seated and all other times is statistically significant ($P < 0.001$). IOP difference between Time 0 in the prone flat versus all other times in the prone flat is statistically significant at every time ($P < 0.05$). IOP difference between Time 0 in prone Trendelenburg and all other times prone Trendelenburg is statistically significant at every time ($P < 0.05$).



did not use bolters nor did we use a Jackson table, so abdominal compression was not minimized in either group. The effect of tetracaine topical anesthetic may have altered the IOP pressure measurements. Finally, we used an applanation tonopen. The most accurate measurement of IOP is invasive monitoring, but this was not ethical in awake subjects.

There are known risk factors for ischemic optic neuropathy, yet determining who will end up with postoperative vision loss is still poorly understood. Practitioners should be aware of risk factors, such as diabetes, aging, chronic hypertension, arteriosclerosis, atherosclerosis, and ischemia.^{26,45–48} They may cause a pathologic change in mean arterial pressure, thereby lowering the IOP needed to decrease perfusion. (perfusion pressure = mean arterial pressure – IOP).^{20,26}

On the other hand, there are unidentifiable risk factors that predispose young healthy patients to ischemic optic neuropathy, such as anatomic variations, watershed zones, and abnormal autoregulation.^{26,34,49}

Precipitating factors in both healthy and sick patients include hypotension, anemia, and increased IOP.²⁶ Combined with predisposing risk factors, these may act as the final injury and result in optic nerve ischemia.

The data herein established IOP increases in the prone Trendelenburg's position, and when combined with other factors, may be a risk factor for postoperative vision loss. Additionally, pressures elevate over time. Although we did not study the reverse Trendelenburg's position, use of a head-up tilt position to decrease IOP has been suggested. Ozcan *et al* discovered that the increase in IOP in prone flat is ameliorated by reverse Trendelenburg.⁵⁰ Similarly, a 15° head-up tilt has been recommended for eye procedures by ophthalmologists.⁴⁵

Based on our study, clinicians may want to consider the following recommendations:

1. Avoid using head down tilt when possible.
2. If Trendelenburg positioning is needed at some point during surgery, be vigilant to return to less risky positions when appropriate.
3. Consider anesthesiologists recording the duration and degree of Trendelenburg used during surgery so that future studies can evaluate complications associated with a head-down position.
4. Ask patients about vision on awakening from surgery, and early ophthalmology referral is warranted if any abnormality exists.^{45,51} Salvage may be possible if underlying causes such as anemia or hypotension are corrected immediately.

The current study may help increase awareness of the mechanism of IOP elevation with surgical positioning and time by filling a specific physiologic knowledge gap. We hope it will stimulate further study of patients in order to better understand risks of IOP elevation. Future studies should look at the effect of prone Trendelenburg on anesthetized patients undergoing surgery.⁵² The amount of amelioration obtained with elevation of the head in reverse Trendelenburg should be included in comparison studies.⁵⁰

■ Key Points

- Prone positioning may risk postoperative vision loss because it increases intraocular pressure which, when combined with other factors, may decrease perfusion pressure to the optic nerve.

- Intraocular pressures are higher in prone Trendelenburg's position than prone flat; therefore, prone Trendelenburg has higher risk.
- Longer time in position increases intraocular pressures, increasing risk.

Acknowledgment

The authors thank Dr. Jason E. Karo, Brooke Army Medical Center, Department of Ophthalmology, for his assistance with this project.

References

1. Myers M, Hamilton S, Bogosian, A, et al. Visual loss as a complication of spine surgery: a review of 37 cases. *Spine* 1997;22:1325–9.
2. Roth S, Barach P. Postoperative vision loss: still no answers yet. *Anesthesiology* 2001;95:575–7.
3. Roth S, Thisted R, Erickson J, et al. Eye injuries after nonocular surgery. *Anesthesiology* 1996;85:1020–7.
4. Cheng M, Sigurdson W, Tempelhoff R, et al. Visual loss after spine surgery: a survey. *Neurosurgery* 2000;46:625–31.
5. Warner ME, Warner MA, Garrity JA, et al. The frequency of perioperative vision loss. *Anesth Analg* 2001;93:1417–21.
6. Manfedini M, Ferrante R, Gildone A, et al. Unilateral blindness as a complication of intraoperative positioning for cervical spine surgery. *J Spinal Disord* 2000;13:271–2.
7. Wolfe S, Lospinus M, Burke S. Unilateral blindness as a complication of patient positioning for spinal surgery. *Spine* 1992;17:600–5.
8. Hoski J, Eismont F, Green B. Blindness as a complication of intraoperative positioning: a case report. *J Bone Joint Surg Am* 1993;75:1231–2.
9. Benumof J, Mazzei W. Multifactorial etiology of postoperative vision loss. *Anesthesiology* 2002;96:1531–2.
10. Bekar A, Tureyen K, Aksoy K. Unilateral blindness due to patient positioning during cervical syringomyelia surgery: unilateral blindness after prone position. *J Neurosurg Anesthesiol* 1996;8:227–9.
11. Grossman W, Ward T. Central retinal artery occlusion after scoliosis surgery with a horseshoe headrest. *Spine* 1993;18:1226–8.
12. Locastro A, Novak K, Biglan A. Central retinal artery occlusion in a child after general anesthesia. *Am J Ophthalmol* 1991;112:91–92.
13. Abraham M, Navin S, Sinha S, et al. Unilateral visual loss after cervical spine surgery. *J Neurosurg Anesthesiol* 15:319–22.
14. Roth S, Nunez R, Schreider B. Unexplained visual loss after lumbar spinal fusion. *J Neurosurg Anesthesiol* 1997;9:346–8.
15. Lee L, Lam A. Unilateral blindness after prone lumbar spine surgery. *Anesthesiology* 2001;95:793–5.
16. Dilger J, Tetzlaff J, Bell G, et al. Ischaemic optic neuropathy after spine fusion. *Can J Anesthesiol* 1998;45:63–6.
17. Katzman S, Moschona C, Dzioba R. Amaurosis secondary to massive blood loss after lumbar spine surgery. *Spine* 1994;19:468–9.
18. Brown R, Schauble J, Miller N. Anemia and hypotension as contributors to perioperative loss of vision. *Anesthesiology* 1994;80:222–6.
19. Stevens W, Glazer P, Kelley S, et al. Ophthalmic complications after spinal surgery. *Spine* 1997;22:1319–24.
20. Dunker S, Hsu H, Sebag J, et al. Perioperative risk factors for posterior ischemic optic neuropathy. *J Am Coll Surgeons* 2002;194:705–10.
21. Lee A. Ischemic optic neuropathy following lumbar spine surgery. *J Neurosurg* 1995;83:348–9.
22. Murphy M. Bilateral posterior ischemic optic neuropathy after lumbar spine surgery. *Ophthalmology* 2003;110:1445–57.
23. Lee LA. ASA Postoperative Vision Loss Registry: preliminary analysis of factors associated with spine operations. *ASA Newsletter* 2003;67:7–8.
24. Ho VT, Newman NJ, Song S, et al. Ischemic optic neuropathy following spine surgery. *J Neurosurg Anesthesiol* 2005;17:38–44.
25. Katz DM, Trobe JD, Cornblath WT, et al. Ischemic optic neuropathy after lumbar spine surgery. *Arch Ophthalmol* 1994;112:925–31.
26. Hayreh S. Anterior ischemic optic neuropathy. *Clin Neurosci* 1997;4:251–63.
27. Setogawa A, Kawai Y. Measurement of intraocular pressure by both invasive and noninvasive techniques in rabbits exposed to head down tilt. *Jpn J Physiol* 1998;48:25–31.
28. Reitsamer H, Kiel J, Harrison J, et al. Tonopen measurement of IOP in mice. *Exp Eye Res* 2004;78:799–804.
29. Mader T, Taylor G, Hunter N, et al. Intraocular pressure, retinal vascular, and visual acuity changes during 48 hours of 10 degrees head down tilt. *Aviat Space Environ Med* 1990;61:810–3.
30. Pillunat L, Anderson D, Knighton R, et al. Autoregulation of human optic nerve head circulation in response to increased intraocular pressure. *Exp Eye Res* 1997;64:737–44.
31. Hargaden M, Goldberg S, Cunningham D, et al. Optic neuropathy following stimulation of orbital hemorrhage in the nonhuman primate. *Ophthalmic Plast Reconstr Surg* 1996;12:264–72.
32. Hayreh S, Weingeist T. Experimental occlusion of the central artery of the retina: retinal tolerance to acute ischemia. *Br J Ophthalmol* 1980;64:896–912.
33. Hargaden M, Goldberg S, Cunningham D, et al. Optic neuropathy following stimulation of orbital hemorrhage in the nonhuman primate. *Ophthalmic Plast Reconstr Surg* 12:264–72.
34. Douthwaite WA. Does the change of anterior chamber depth or/and episcleral venous pressure cause intraocular pressure changes in postural variation? *Optom Vis Sci* 1997;74:664–7.
35. Friberg T, Sanborn G, Weinreb R. Intraocular and episcleral venous pressure increase during inverted posture. *Am J Ophthalmol* 1987;103:523–6.
36. Draeger J, Hanke K. Postural variation of intraocular pressure: preflight experiments for the D1 mission. *Ophthalmic Res* 1986;18:55–60.
37. Cheng M, Todorov A, Tempelhoff R, et al. The effect of prone positioning on intraocular pressure in the anesthetized patient. *Anesthesiology* 2001;95:1351–5.
38. Hunt K, Bajekal R, Calder I, et al. Changes in intraocular pressure in anesthetized prone patients. *J Neurosurg Anesthesiol* 2004;16:287–90.
39. Lam A, Douthwaite W. Does the change of anterior chamber depth or/and episcleral venous pressure cause intraocular pressure changes in postural variation? *Optom Vis Sci* 1997;74:664–7.
40. Mansour A, Feghali J, Jaroudi N. Increased intraocular pressure with head down position. *Am J Ophthalmol* 1984;98:114–5.
41. Cook J, Friberg T. Effect of inverted body position on intraocular pressure. *Am J Ophthalmol* 1984;98:784–7.
42. Friberg T, Weinreb R. Ocular manifestations of gravity inversion. *JAMA* 1985;253:1755–7.
43. LeMarr J, Golding L, Adler J. Intraocular pressure response to inversion. *Am J Optometry Physiol Optics* 1984;61:679–82.
44. Tsamparlis J, Casey T, Howell W, et al. Dependence of intraocular pressure on induced hypotension and posture during surgical anesthesia. *Trans Ophthalmol Soc UK* 1980;100:521–6.
45. Williams E. Postoperative blindness. *Anesthesiol Clin North Am* 2002;20:605–22.
46. Connolly S, Gordon K, Horton J. Salvage of vision after hypotension-induced ischemic optic neuropathy. *Am J Ophthalmol* 1994;117:235–42.
47. Haeflinger I, Meyer P, Flammer J, et al. The vascular endothelium as a regulator of the ocular circulation: a new concept in ophthalmology? *Surv Ophthalmol* 1994;39:123–32.
48. Hayreh S, Podhajsky P, Zimmerman B. Nonarteritic anterior ischemic optic neuropathy: time of onset of visual loss. *Am J Ophthalmol* 1997;124:641–7.
49. Hayreh S. In vivo choroidal circulation and its watershed zones. *Eye* 1990;4:273–89.
50. Ozcan M, Praetel C, Bhatti T, et al. The effect of body inclination during prone positioning on intraocular pressure in awake volunteers: a comparison of two operating tables. *Anesth Analg* 2004;99:1152–8.
51. Williams E. Postoperative ischemic optic neuropathy. *Anesth Analg* 1995;80:1018–29.
52. Jantzen J, Hennes H, Rochels, R, et al. Deliberate arterial hypotension does not reduce intraocular pressure in pigs. *Anesthesiology* 1992;77:536–40.